

CLINICAL VIGNETTE

Diabetic Amyotrophy Presenting as Weight Loss, Foot Drop and Truncal Pruritus

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A 63-year-old male without primary care for over 10 years and recently diagnosed type 2 diabetes presented to establish care and for follow-up on his diabetes. Two months ago he was diagnosed by an outside physician with a HgbA1c of 9.8% and initially started on sitagliptin- metformin combination. He was able to transition to metformin alone after starting diet and exercise program with rapid improvement of his HgbA1c to 5.9% over two months.

At his initial visit, he described a plethora of symptoms that accompanied his recent diagnosis of diabetes. Over the prior 8 months, he reported unintentional weight loss of 30 lbs, ongoing fatigue, loss of appetite, and right foot numbness and weakness which led to a pronounced foot drop. Over the past month, he developed severe truncal pruritus localized to his left thigh and waist areas, without any visible rash.

Broad testing was initiated. Labs were notable for an elevated ferritin level to 786 ng/mL, low vitamin B12 of 235 pg/mL, normal folate level 9.6 ng/mL, bilirubin <0.2mg/dL, chemistries, PSA, ESR, CRP and TSH were normal and SPEP was without monoclonal bands. HIV, Hepatitis C antibody, and RPR were also negative. Lumbar sacral electromyogram and nerve conduction study revealed a sensorimotor axonal polyneuropathy as well as likely subacute right L5 nerve root dysfunction. This study did not include thoracic nerves. MRI of the lumbar spine showed S1 perineural root cysts, felt unlikely to be the cause of the patient's neurologic findings. CT scans of the chest, abdomen and pelvis were unrevealing. Colonoscopy was also normal. He was referred to physical therapy and fitted with a right ankle-foot orthosis to improve ambulation.

Over the next six months, the patient's pruritus persisted, which was described as a "living hell" without visible rash. The itching was worse with movement, warmth, and stress. He tried stopping all of his current medications, without any improvement in his symptoms. Several medications were prescribed to alleviate the itching, including hydrocortisone, triamcinolone cream, topical capsaicin, oral antihistamines, and diphenhydramine, all with only minimal benefit. He was seen by both dermatology and allergy without any clear diagnosis or further recommendations. He is currently receiving acupuncture therapy and taking gabapentin for treatment of his neuropathy.

Discussion

Diabetic neuropathy is the most common complication of diabetes affecting ~50% of individuals living with diabetes. Diabetic neuropathy is frequently a distal symmetric polyneuropathy (glove-and-stockings) and autonomic neuropathy.¹ However, diabetic neuropathy is heterogeneous and may manifest less commonly as isolated or multiplex mononeuropathy or lumbosacral, thoracic, or cervical radiculopathy.^{1,2}

Diabetic amyotrophy is most well-known form of diabetic radiculopathy affecting ~1% of diabetics.^{3,4} Consistent with our patient, diabetic lumbar radiculopathy tends to affect middle aged males, with type 2 diabetes of variable duration and glycemic control.³⁻⁵ Individuals initially develop symptoms of unilateral pain accompanied by weakness, foot drop and proximal leg muscle atrophy.³⁻⁵ Like our patient, individuals frequently experience significant unintended weight loss which can prompt evaluation for occult malignancy.³⁻⁵ About half will experience autonomic symptoms.⁵ Finally, as the disease progresses, other nerve roots may be affected and there can be bilateral involvement. Pertinent to our case, 12% will develop concurrent thoracic radiculopathy and we believe this likely explains the intense pruritus on the contralateral trunk experienced by our patient.⁶

Typical diabetic neuropathy (distal symmetric polyneuropathy) can be diagnosed and managed in the primary care setting with careful foot exam including monofilament, vibration and ankle reflex testing as well as lab evaluation for secondary causes which may include TSH, B12, folic acid, SPEP, CBC and CMP.^{1,7,8} In contrast, atypical diabetic neuropathy has acute or subacute onset, asymmetry, non-length dependent, with motor predominant, and/or multifocal. Radiculopathy should be referred to neurology for consultation and EMG testing.^{1,7,8} Diagnosis of diabetic lumbar radiculopathy is supported by EMG abnormalities and elevated inflammatory markers such as elevated ESR as well as an otherwise negative secondary workup for other etiologies.^{3,4} Our patient was found to have localized EMG abnormalities consistent with his diagnosis of lumbar radiculopathy and otherwise negative work up for secondary cause supporting diagnosis of diabetic lumbar radiculopathy despite his normal ESR. Lumbar puncture to assess for elevated CSF protein and nerve biopsy to document microvasculitis may rarely be required but remain a diagnostic option in uncertain presentations.⁵ These were not necessary in this case.

Current management is supportive focusing on pain control and rehabilitative therapies to address muscle weakness. While there is suggestive evidence that diabetic lumbar radiculopathy may be secondary to ischemic microvasculitis, there is insufficient evidence to recommend immunotherapy therapy.^{4,9} However, steroids could be considered for symptomatic relief in refractory cases.⁹ Individuals with diabetic lumbar radiculopathy show improvement over months to a year but recovery is often partial.³

Conclusions

Lumbosacral radiculopathy (diabetic amyotrophy) is the most common diabetic radiculopathy encountered in the clinical setting. Incidence is not dependent on duration of diabetes or severity of current glucose control. While symptoms are typically unilateral, the disease may progress to bilateral involvement and to the thoracic nerve roots. Clinicians should include diabetic lumbar radiculopathy in their differential when diabetic individuals present with unintended weight loss and asymmetric neuropathic symptoms.

REFERENCES

1. **Pop-Busui R, Boulton AJ, Feldman EL, Bril V, Freeman R, Malik RA, Sosenko JM, Ziegler D.** Diabetic Neuropathy: A Position Statement by the American Diabetes Association. *Diabetes Care*. 2017 Jan;40(1):136-154. doi: 10.2337/dc16-2042. PMID: 27999003; PMCID: PMC6977405.
2. **Tesfaye S, Boulton AJ, Dyck PJ, Freeman R, Horowitz M, Kempler P, Lauria G, Malik RA, Spallone V, Vinik A, Bernardi L, Valensi P; Toronto Diabetic Neuropathy Expert Group.** Diabetic neuropathies: update on definitions, diagnostic criteria, estimation of severity, and treatments. *Diabetes Care*. 2010 Oct;33(10):2285-93. doi: 10.2337/dc10-1303. Erratum in: *Diabetes Care*. 2010 Dec;33(12):2725. PMID: 20876709; PMCID: PMC2945176.
3. **Albers JW, Pop-Busui R.** Diabetic neuropathy: mechanisms, emerging treatments, and subtypes. *Curr Neurol Neurosci Rep*. 2014 Aug;14(8):473. doi: 10.1007/s11910-014-0473-5. PMID: 24954624; PMCID: PMC5084622.
4. **Tracy JA, Dyck PJ.** The spectrum of diabetic neuropathies. *Phys Med Rehabil Clin N Am*. 2008 Feb;19(1):1-26, v. doi: 10.1016/j.pmr.2007.10.010. PMID: 18194747; PMCID: PMC2720624.
5. **Llewelyn JG.** The diabetic neuropathies: types, diagnosis and management. *J Neurol Neurosurg Psychiatry*. 2003 Jun;74 Suppl 2(Suppl 2):ii15-ii19. doi: 10.1136/jnnp.74.suppl_2.ii15. PMID: 12754324; PMCID: PMC1765622.
6. **Pasnoor M, Dimachkie MM, Barohn RJ.** Diabetic neuropathy part 2: proximal and asymmetric phenotypes. *Neurol Clin*. 2013 May;31(2):447-62. doi: 10.1016/j.ncl.2013.02.003. Epub 2013 Mar 15. PMID: 23642718; PMCID: PMC4183450.
7. **Feldman EL, Callaghan BC, Pop-Busui R, Zochodne DW, Wright DE, Bennett DL, Bril V, Russell JW, Viswanathan V.** Diabetic neuropathy. *Nat Rev Dis Primers*. 2019 Jun 13;5(1):41. doi: 10.1038/s41572-019-0092-1. PMID: 31197153.
8. **Watson JC, Dyck PJ.** Peripheral Neuropathy: A Practical Approach to Diagnosis and Symptom Management. *Mayo Clin Proc*. 2015 Jul;90(7):940-51. doi: 10.1016/j.mayocp.2015.05.004. PMID: 26141332.
9. **Llewelyn D, Llewelyn JG.** Diabetic amyotrophy: a painful radiculoplexus neuropathy. *Pract Neurol*. 2019 Apr;19(2):164-167. doi: 10.1136/practneurol-2018-002105. Epub 2018 Dec 8. PMID: 30530723.